In the pediatric literature, copper deficiency is widely reported [1–10]. Hereditary hypoceruloplasminemia [11] and hypocupremia with neurological abnormalities [12] are also reported among genetic abnormalities. Nevertheless, our patient’s symptoms cannot be attributed to those diseases or to familial benign copper deficiency [14], because he had dark hair, hypoceruloplasminemia and severe anemia. We also want to underline that his iron serum level, which was pathologically high before copper supplementation, decreased to a normal range after therapy. Nevertheless, even if the value had fallen, this could be due to iron incorporation into red blood cells for erythropoiesis recovery: ferrokinetic studies on pediatric patients showed an increased bone marrow activity with improvement in iron utilization after copper supplementation: serum iron can fall to a low level, so that a supplemental iron therapy can be required [3]. On the other hand, the hypotheses about iron mobilization in copper deficiency are widely confirmed by authors [3, 13]. Finally, it is known that in Wilson’s disease the liver becomes unable to bind copper to ceruloplasmin; so nonceruloplasminic copper level increases and ceruloplasminic copper level and ceruloplasmin level decrease [15]. On the other hand, it is also reported that serum copper level is elevated in pregnancy, lymphomas, infections and other diseases, showing that copper serum level does not suitably reflect tissue level [1, 16]. Moreover, evaluations on pediatric patients, though very interesting, cannot be a model for adults, because it is known that total body copper concentration in children is higher than in adults and copper distribution is very different [1, 2]. Thanking for the interesting observations, we refer the kind correspondent to the bibliography of our paper, from which we have drawn these remarks.

References
